The Journal

of the

Louisiana State Medical Society

Vol. 106, No. 4
$4.00 Per Annum, 35c Per Copy

APRIL, 1954

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

MEDICAL MANAGEMENT OF PATIENTS WITH URINARY STONE

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The medical management of stone-forming patients is important because of the well known tendency to recurrence in 15 to 20 per cent of all cases. Unfortunately, our stone prevention programs have not been too successful. As a result of the study of calculous disease by new techniques for the past fifteen years, it has been possible to obtain new data on the stone forming process which may be of value in prevention of recurrence. The techniques which were used in this study were borrowed from mineralogy and are called polarization optics and x-ray diffraction photography. Since urinary calculi are crystalline it is possible to study them with the techniques used to study the crystals of minerals. With these methods it is possible to accurately identify crystalline material, even in microscopic amounts. With the petrographic (polarizing) microscope it is also possible to study intimately the relationship of such crystalline material to its surroundings, even when in situ in the tissue of the kidney. This instrument makes it possible to analyze all portions of a urinary calculus, including the nucleus and the various layers, and to learn something of the manner in which it was formed. It is believed that the first portion of the stone formed, called the nucleus, is of causal significance. This will be discussed later. The nucleus may usually be found only by optical study. The techniques used, the descriptions of the various crystalline components of calculi, and the clinical implications of these findings have been reported in previous papers [1, 2].

This paper concerns itself mainly with the details of management of patients who have calcium-containing calculi, comprising approximately 90 per cent of all cases of urolithiasis in North America. There are probably only 3 important crystalline substances in calcium-containing calculi—calcium oxalate, calcium phosphate, and magnesium ammonium phosphate. The presence of the third substance in a calculus is almost always due to urea-splitting infection.

PRIMARY AND SECONDARY CALCIUM-CONTAINING CALCULI

The calcium-containing calculi fall naturally into two groups on a clinical basis. The distinction seems to be based mainly on urinary reaction. Primary calcium-containing calculi occur in individuals with an acid urine in an apparently normal urinary tract and are usually composed of calcium oxalate or of mixed calcium oxalate and calcium phosphate. If infection is present it is by organisms which do not render the urine alkaline.

The other group of calcium-containing calculi are generally considered "secondary" calculi,—secondary to or associated with other urinary tract pathology, such as obstruction, stasis, or chronic infection. The urine is almost invariably alkaline due to the presence of urea-splitting organisms. These stones are generally com-

* Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

posed of magnesium ammonium phosphate and calcium phosphate—and are exemplified by the common dendritic or "staghorn" stone.

This classification is summarized in Table 1.

TABLE 1
CONDITIONS OF OCCURRENCE OF CALCIUM-CONTAINING CALCULI

Content	Environment
Calcium oxalate or Calcium oxalate-calcium phosphate	Normal urinary tract, urine acid usually, infection + or —
$MgNH_4PO_4 \cdot CaPO_4$	Abnormal urinary tract, alkaline urine, urea-splitting infection

However, all calculi which are of the "staghorn" or dendritic type are not associated with urea-splitting infection. About 20 per cent of them are composed of almost pure calcium phosphate, and occur in a urine most commonly slightly acid.

As a result of infection by urea-splitting organisms a primary stone of any composition may acquire an external portion of calcium phosphate and magnesium ammonium phosphate and have the external appearance of an "alkaline infection" stone. It is unwise to guess at the composition of a stone from its external appearance. Color is also an unreliable guide. I have seen calcium oxalate, calcium phosphate, and uric acid stones all of identical color and texture.

We commonly divide calcium-containing calculi into "oxalate" and "phosphate" stones, implying that the "phosphate" calculi are secondary stones associated with urea-splitting infection. This is not always true, either from the standpoint of pathogenesis or treatment. All phosphate stones do not occur in an alkaline urine but some may occur in an acid urine with no urea-splitting infection being present (as we have just pointed out). Furthermore, many of the primary "oxalate" stones contain large amounts of phosphate. In fact, many of these so-called "oxalate" stones originated as "phosphate" stones because the nucleus is of calcium phosphate.

The noncalcium containing calculi are composed of uric acid and cystine. Usually (but not always) these are "pure" calculi. They occur in acid urine, usually uninfected (except secondarily).

THE NUCLEUS

A nucleus in physical chemistry is that part of a concretion which is formed first and upon which further precipitation occurs to produce growth of the concretion. Optical study has shown that many calculi have a demarcated portion, often of different composition or structure than the rest of the stone, and so situated that it must represent the first portion of the stone formed.

Theoretically, the nucleus should be a very minute structure—the first particle deposited from solution. In calculi the term is applied to a larger structure, varying from microscopic size to several millimeters in its dimensions. Obviously, this larger "nucleus" is an extension of the growth of the first particle deposited from solution and is of the same composition.

There are two ways in which nuclei are formed:

1. From Randall's plaques [3] situated upon the renal papilla. Deposits of crystalline material, most commonly of calcium phosphate, occur beneath the epithelium of the renal papilla. When, by accident of position, this submucosal plaque erodes to the calyceal surface, it becomes exposed to the supersaturated urine of the calyx. Precipitation of urinary salts upon this nucleus produces an asymmetrically developed stone with the nucleus upon the external surface of the stone. (Fig. 1)

2. The second way in which nuclei are formed is by precipitation of urinary salts in the urine itself (not upon a mucosal surface as above). Such a nucleus occurs in the center of a symmetrically developed stone. (Fig. 2)

The nuclei of the great majority of calcium-containing calculi are composed of calcium phosphate and this applies also to the nuclei of calcium oxalate calculi. A detailed consideration of the nucleus appears in a previous paper [2].

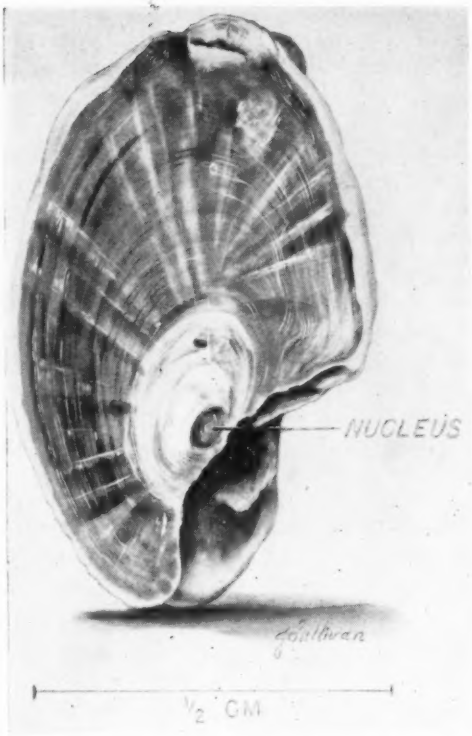

Figure 1.—Asymmetrically developed calcium oxalate calculus with a calcium phosphate nucleus upon the external surface of the stone. Develops from a Randall's plaque upon the mucosa of the renal papilla.

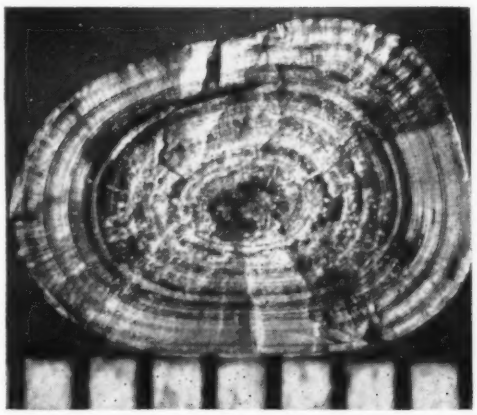

Figure 2.—Symmetrically developed uric acid calculus with a central nucleus of uric acid. Originates in urine by simple precipitation from solution.

SUPERSATURATION OF URINE

Meyer[4] found that normal urine is commonly supersaturated 2 to 4 times with some of the stone-forming salts at all *p*H levels without precipitation occurring. It is able to retain these salts in such a supersaturated state *for a time if no nucleus is present.* If a nucleus *is* present precipitation may occur whenever the concentration exceeds the saturation point. So that, whereas the average person may be able to keep his urine supersaturated 2 to 4 times with the various urinary salts, the person with a nucleus present in the urinary tract may precipitate salts whenever the saturation point is exceeded. It would seem desirable, therefore, to conserve this power of urine to hold its salts in supersaturated solution by preventing nuclei from forming.

Meyer found that: (Fig. 3)

1. The average amount of uric acid present in normal urine supersaturates it

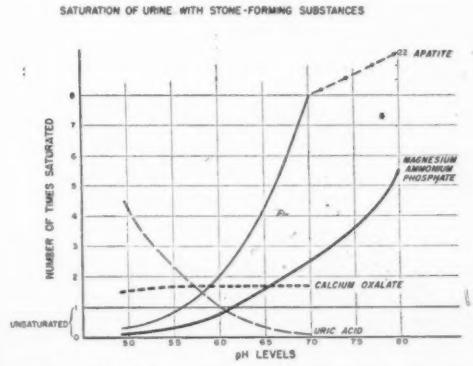

Figure 3.—Saturation of urine by the average amount of each of the common stone-forming salts present in normal urine, according to J. Meyer. Normal urine is able to maintain its salts in a 2 to 4 times supersaturated solution for a time without precipitation occurring if no nucleus is present. In the presence of a nucleus precipitation may occur when the saturation point is exceeded. (Apatite is calcium phosphate).

4 times at *p*H 5.0 but unsaturates it above *p*H 6.0. This means that the solubility of uric acid increases with increasing alkalinity. So we alkalinize the urine to prevent uric acid stone. The same applies to cystine.

2. The average amount of calcium oxa-

late present in normal urine supersaturates it 1.7 times and the solubility is practically unchanged by changing the *p*H. Therefore, acidification or alkalinization of the urine is useless in treatment to prevent calcium oxalate stone.

3. The average amount of calcium phosphate present in normal urine supersaturates it 8 times at *p*H 7.0, supersaturates it 4 times at *p*H 6.5, supersaturates it only 2 times at *p*H 6.0 and unsaturates it below *p*H 5.6. Note that the solubility of this average amount of calcium phosphate approximately doubles with each half unit fall in *p*H. This is a very substantial increase in solubility in a narrow *p*H range. Of course, stone-forming patients often have increased amounts of calcium phosphate present in the urine; solubility of these increased amounts of calcium phosphate will not be doubled under these circumstances but it will be increased.

Similar values obtain for magnesium ammonium phosphate but they are less remarkable than is the case with calcium phosphate.

So that we acidify the urine to increase the solubility (decrease the degree of saturation) of calcium phosphate and magnesium ammonium phosphate.

UREA-SPLITTING INFECTION

With the onset of urea-splitting infection marked alkalinity of the urine occurs and calcium phosphate and magnesium ammonium phosphate are precipitated in increasing amounts. The marked insolubility of these two substances in alkaline urine emphasizes the necessity of eradicating urea-splitting infection if we are to prevent recurrence of such calculi. This is often a difficult problem, even in these days of potent antibiotics. If the *p*H cannot be lowered stone recurrence will usually follow.

HYPERCALCURIA

Flocks [5], Albright [6] and others have shown that a relationship exists between the amounts of calcium present in the urine and the incidence of calcium stone. The amount of calcium excreted may be increased by excessive ingestion of calcium-containing foods or calcium salts, excessive ingestion of vitamin D, hyperparathyroidism, acidosis (as of ammonium chloride therapy), disuse atrophy of the bony skeleton caused by prolonged immobilization of fractures, muscular paralysis or debilitating disease, postmenopausal osteoporosis, Cushing's syndrome, and sarcoid.

Excessive Ingestion of Calcium in Food and Medication.—Milk is the main source of calcium in our diet. Many persons drink large quantities of milk because it is a "perfect" food or because it is indicated in peptic ulcer; the etiologic relationship to stone is obvious. The use of calcium salts for peptic ulcer is open to the same objection. The administration of large amounts of vitamin D for rickets, tetany and rheumatoid arthritis will produce hypercalcuria. Happily this form of therapy is no longer popular.

Hyperparathyroidism. — Hyperparathyroidism is the cause of about 5 per cent of urinary calculi. In this condition the blood serum calcium is elevated and the blood serum phosphorus depressed. However, both the urinary calcium and phosphorus are increased.

Commonly a blood serum calcium level in excess of 10.5 mg. per 100 cc. and a blood serum phosphorus level below 3.0 mg. per 100 cc. should raise the suspicion that this disease may be present. One should place more reliance on a low serum phosphorus level than on a high serum calcium level because the test for phosphorus is easy and reliable while the test for calcium is often unreliable. In a patient with calculi the criteria for making the diagnosis of hyperparathyroidism are, in order of importance: (1) a serum phosphorus below 3 mg., (2) hypercalcuria in excess of 150 mg. per day, and (3) an elevated serum calcium.

It has been my observation that physicians tend to rely almost entirely upon the serum calcium and phosphorus levels in making the diagnosis of hyperparathyroidism to the almost virtual exclusion of urinary calcium studies. We find it useful to

do twenty-four hour quantitative urinary calcium determinations in borderline cases with the patients on a standard low calcium diet and regard a urinary calcium excretion of 150 mg. per day as the upper limit of normal.

We also find that the use of the Sulkowitch reagent is valuable as a rough bedside or office test to determine whether hypercalcuria is present or not. If the test shows that little calcium is present on several determinations it is very unlikely that hyperparathyroidism is present. On the other hand, if the test repeatedly shows large amounts of calcium one has to be cautious about ruling out hyperparathyroidism, even if the first serum calcium and phosphorus levels are within normal limits (from Albright).

Acidosis.—Acidosis as a cause for hypercalcuria must receive consideration because acidification therapy is used in prevention of calcium-containing calculi. Ammonium chloride and acid-ash diets are commonly prescribed to acidify the urine because calcium phosphate and magnesium ammonium phosphate are more soluble in an acid urine. Acidification therapy may produce acidosis with consequent increased rate of dissolution of bone and resultant hypercalcuria. Under these circumstances any benefit achieved by increased solubility of calcium phosphate may be partially or completely nullified by increased excretion of the same salt.

Disuse Atrophy of Bone.—The demineralization of bone in immobilized patients leads to the so-called recumbency calculi. This demineralization may be mitigated to some extent by frequent changes in position of bedridden patients, by bed exercises, and early ambulation. The services of a physiotherapist are invaluable in this regard. Do not give large amounts of milk to immobilized fracture patients because of the mistaken idea that a large amount of calcium in the diet is necessary to heal a fracture; it only leads to stone formation.

Idiopathic Hypercalcuria.—Flocks found that many patients with calcium urolithiasis "have a high urinary calcium excretion without any bone disease, evidence of hyperparathyroidism, change in the blood calcium or phosphorus levels, or other demonstrable abnormality of calcium metabolism." Multiple calculi may be present, often limited to one kidney. The kidneys may appear normal by pyelographic study and there may be no discernible diminution in renal function. Albright and Reifenstein [6] find that many of these cases have or have had a staphylococcal pyelonephritis. Prognosis is guarded; about half will recover completely (private communication). The mechanism may be some form of renal tubular damage.

PRINCIPLES—MEDICAL MANAGEMENT TO PREVENT RECURRENCE

The underlying principle in prevention of stone is to prevent the excess supersaturation of the urine by substances which might precipitate to form stone. We will exclude, for the moment, the intangibles of vitamins and colloids but will mention them later. There are three ways to reduce the degree of saturation of the urine with stone-forming salts: (1) by increasing urinary volume to dilute the concentration of salts; (2) by decreasing urinary excretion of the offending crystalloid; and (3) increasing urinary solubility of the offending crystalloid.

Forcing Fluids.—Forcing of fluids is the most reliable method of reducing urinary supersaturation. The nuclei of calculi are small and may form in a short time in a concentrated urine. With this in mind it is my feeling that the statement to "force fluids" must be amplified by instructions to the patient to prevent even transient dehydration *in the patient who is a recurrent stone former.* This must be stated in terms understandable to the patient. To merely tell a patient to drink a certain quantity each day is not enough. The recurrent stone former must be told to spread his fluid intake over his waking hours, including some fluids in the evening even if it results in some nocturia. Of course, the instruction of the patient should be tempered by the gravity of the

situation. More cooperation may be expected from the patient who has had considerable trouble in the past.

MEDICAL MANAGEMENT: CALCIUM-CONTAINING CALCULI

Specific therapy to prevent recurrence of calcium-containing calculi consists mainly in keeping as much calcium out of the urine as possible and in keeping in a dissolved state those phosphate salts which do get into the urine. This is accomplished by combinations of diet and acid salt therapy which decrease calcium excretion in the urine and render the latter more acid, and by the control of urea-splitting infection. While it has not been conclusively shown that high oxalate foods contribute to calcium oxalate stone formation, avoidance of considerable amounts of such foods would seem justified. It is understood, of course, that other well-recognized measures of therapy, such as surgical removal of urinary tract obstructions, treatment of all urinary infections (in addition to urea-splitting infection), and treatment of other relevant lesions, such as foci of infection, outside the urinary tract, must also be carried out.

Specific Treatment of Calcium Oxalate Calculi.—It has been generally assumed that there is little we can do to prevent recurrence of calcium oxalate stone except to force fluids. However, crystallographic study has shown that calcium phosphate occurs as the nucleus in the majority of stones of predominant calcium oxalate composition. Calcium phosphate is increasingly soluble in acid urine. Because the absence of a nucleus may permit the urine to maintain its dissolved calcium oxalate in supersaturated solution more readily, it is hereby suggested that we treat calcium oxalate stone-formers by acidification therapy to prevent the calcium phosphate nuclei from forming. It is fair to state that the validity of this proposition has not been tested upon any long series of patients.

Calcium oxalate stone-formers should refrain from eating large amounts of food high in oxalate even though it has not been demonstrated that high oxalate foods are a factor in stone formation. (See list in Treatment Summary at end of paper). While tea and coffee also contain considerable amounts it is to be remembered that we do not eat these substances but only drink a beverage made by heating them in water.

Low Calcium Diet.—Milk is the most abundant source of calcium in the American diet. In general, I have been content to severely restrict the use of milk and its products, except for butter, in patients with hypercalcuria and to pay little attention to other calcium-containing foods. This simple restriction is easy to remember and no patient should complain that such a regimen is arduous. It should be obvious that cream, cheese, condensed milk, buttermilk, ice cream, cream soups, milk gravies, cheese souffles, Welsh rarebits, and custards, are included in the forbidden list. Butter contains little calcium and may be used freely. On such a diet the intake of calcium will not exceed 150 mg. daily.

Occasionally, a stone-former will be discovered who has some peculiar dietary habit. We found one woman who ate a large amount of molasses on bread daily. Molasses is a high calcium food with a very high alkaline ash and, therefore, doubly undesirable. We also found a cook in a restaurant who passed 11 stones in two years and who "nibbled" on handsful of olives, raisins, shelled nuts and slabs of cheese, (all high calcium foods), daily at his work. The average person eats no molasses at all and his intake of such foods as olives, raisins and nuts is so low that they need not be included in a routine list of forbidden foods. There is nothing so frustrating to the average stone-forming patient as a long list of forbidden foods, most of which he rarely eats. However, it is a good idea to run through the list of high calcium foods at the end of this paper with the patient; if it appears that any of them are eaten in large amounts their use should be reduced to "normal" amounts. In some high calcium foods the

calcium is combined with oxalate; it is extremely doubtful that the calcium will be absorbed when so combined.

Acidification of Urine.—The solubility of calcium phosphate and magnesium ammonium phosphate increase with increase in acidity of the urine. Higgins [7] first called attention to the possibilities of acidification therapy by dietary means to prevent urinary calculi. By the use of acid ash diets he has been able to prevent recurrence of phosphatic calculi and to produce dissolution of some renal calculi in situ. He has stressed that such treatment does not always work. Many workers have had little success with this method. It may be pointed out that it is quite feasible to produce resorption of a small calyceal calculus when it is composed of calcium phosphate. If the calcium phosphate nucleus has acquired an external layer of calcium oxalate much less should be expected from attempts at resorption by producing an unsaturated urine; calcium oxalate is resorbed with difficulty.

In general, meats and cereals promote an acid reaction in the urine while fruits, vegetables and milk produce an alkaline reaction. There are numerous exceptions; for instance, cranberries, plums, and prunes render the urine acid. The consumption of orange juice, grapefruit juice and tomato juice should be investigated if one is attempting to produce an acid urine; many people consume large amounts of these juices. Although they are only weakly alkaline ash foods an excessive consumption might pile up an excess of alkaline ash radicles which would be undesirable. Keep the intake of these juices down to a reasonably low level. Check over the list of high alkaline ash foods in the summary at the end of the paper to be sure that your patient does not consume large amounts of any one.

It has been the writer's experience that most patients will not remain on stringent diets for a long time but are quite willing to take oral medication. Where such "pill swallowing" is without demonstrable harmful effects and accomplishes the desired result it is my feeling that it is to be preferred to severe restriction in diet, for the practical reason that it may result in longer cooperation on the part of the patient. The combination of mild dietary restriction plus harmless acidifying salt therapy is worthy of a trial in many patients; it is not indicated in all.

Ammonium chloride, acid ash diet, and sodium acid phosphate have been used to acidify the urine. The first two are open to the objection that they may produce acidosis and bone demineralization and may increase the excretion of calcium phosphate in the urine.

Cordonnier and Talbot [8] have found that sodium acid phosphate is the drug of choice in patients with calcium-containing calculi. It is a moderately effective acidifier. In addition, it reduces urinary calcium excretion by depressing serum calcium levels slightly. There may be an increase in urinary phosphorus excretion but it is mostly in the form of soluble sodium acid phosphate. Administration of sodium acid phosphate will not produce acidosis or bone demineralization, even if used in large dosage over a long period.

There has been no apparent change in the serum calcium levels of any of the patients taking sodium acid phosphate, either in my own series or that of Cordonnier (private communication). The customary dosage of sodium acid phosphate is 0.6 gm. (10 grains) four times daily. On this dosage Cordonnier was able to reduce urinary calcium excretion 50 per cent both in ambulatory and recumbent patients.

A possible objection to the use of sodium acid phosphate occurs in urea-splitting infection. In this condition the use of the salt may throw an added amount of phosphate into the urine, presumably in an insoluble form as magnesium ammonium phosphate, to increase the already excessive deposition of that salt. In acid urine the phosphate occurs in solution as sodium acid phosphate which is quite soluble.

Determination of Urinary pH.—Urinary *p*H is determined with a liquid colorimet-

ric indicator, (the LaMotte *p*H Indicator*). This is quite superior to most of the paper indicators because it gives a definite color change for each 0.5 unit change in *p*H, from a scarlet red at *p*H 4.5 to a blue green at *p*H 8.0 The writer has had some difficulty in determining half unit changes in *p*H with nitrazine paper.

Determination of Urinary Calcium.—The Sulkowitch test is used for rough quantitative determination of urinary calcium in the office or at the bedside. A bottle of the reagent should stand on the same shelf with reagents used to determine urinary albumin and sugar. The test is very simply and quickly done.

The Sulkowitch reagent is made as follows: oxalic acid 2.5 gm., ammonium oxalate 2.5 gm., glacial acetic acid 5.0 cc., distilled water q. s. ad 150 cc. A calcium determination is made by adding 2 cc. of the reagent to 5 cc. of urine in a test tube and noting the result. If no calcium is present the urine will remain clear. If calcium is present it will be precipitated as calcium oxalate, forming a white cloud or turbidity whose density will vary with the amount of calcium. The density of the turbidity should be recorded as 0 or 1 to 3 +. It is necessary to take into consideration the specific gravity of the urine in evaluating the Sulkowitch test. One should familiarize himself with the range of results possible with this test by using it routinely on a series of patients, including non-stone formers, for a while before attempting to interpret results.

Normal urines may show but slight turbidities; these are classed as 1 +. Moderate to marked turbidity represents hypercalcuria of grades 2 or 3, making due allowance, of course, for the concentration or dilution of the urine. A strongly positive 2 or 3 + Sulkowitch test will suggest hyperparathyroidism and make further study necessary. If repeated studies rule out this disease one looks for another cause or thinks of "idiopathic hypercalcuria."

Details of pH and Calcium Control.—It should be pointed out that determinations of urinary calcium and *p*H upon a single urine are of limited value in a stone prevention program because of the variability of these values in most individuals. Reliance should only be placed upon determinations made on several urines taken at different times of the day and this should be repeated at intervals. Until a treatment regimen has been stabilized the patient should be seen as often as twice weekly. At each visit the patient is asked to bring along several specimens collected at various times of the day, including the first urine in the morning.

An attempt should be made to keep the *p*H of the urine as low as possible in patients with calcium-containing calculi. The normal urine is unsaturated with calcium phosphate below *p*H 5.6 and one should strive to approach this *p*H value. Any significant decrease in *p*H accomplished on the acid side of neutrality is worthwhile, as was pointed out in an earlier paragraph. It will ordinarily be impossible to acidify the urine in the presence of urea-splitting infection, or in patients who regularly take alkalies or milk of magnesia.

If the *p*H cannot be brought down lower than 7.0 greater emphasis must be placed upon the two other methods of decreasing the degree of supersaturation of the urine by stone-forming salts. These are (1) forcing of fluids to increase the urinary volume (and thus dilute the concentration of the salts), and (2) restriction in the intake of calcium in the diet. It should be pointed out that the former is always of value while the latter often fails.

Careful maneuvering by adjustment of acidification, dietary calcium, and fluid intake may sometimes so reduce the supersaturation of the urine that precipitation and consequent stone-formation may be inhibited. I am very conscious of the fact

*The LaMotte Reaction Outfit is obtainable from the LaMotte Chemical Co., Towson, Baltimore 4, Maryland.

that the desired result is not always achieved.

Chronic Infection with Stone.—In certain patients who have some degree of renal insufficiency with chronic changes (parenchymal scarring, dilated atonic pelves and ureters), it is manifestly impossible to eradicate urea-splitting infection or to acidify the urine. Sodium acid phosphate will be useless as an acidifier, and may be contraindicated.

Shorr [9], in 1945, proposed the use of aluminum hydroxide jel (amphojel), and more recently basic aluminum carbonate jel (basaljel),[10] to reduce the amount of phosphorus absorbed from the bowel, and thus to reduce the amount excreted by the kidneys. The phosphorus is converted to an insoluble aluminum phosphate in the intestine and excreted in the stool. A low phosphorus diet is also given. Shorr has pointed out that impaired renal function and urinary infection do not hamper the effectiveness of this form of therapy. It may be used in uremia. Marshall [11] has reported favorable results, especially in patients who show a marked tendency to stone recurrence associated with intractable infection and chronic changes in the urinary tract. Nephrectomy should be considered in patients with severe *unilateral* lesions of this type. The basaljel-low phosphorus diet regimen has also been used with success in the prevention of recumbency calculi.

In my own experience the amount of any of these aluminum jels necessary daily has posed a problem for patients with sensitive stomachs; it may fairly be said that these compounds are unpleasant to take. Under the circumstances, a high degree of cooperation will be difficult to obtain except in patients who have a very serious stone situation.

MEDICAL MANAGEMENT: URIC ACID AND CYSTINE CALCULI

The application of isotope techniques has shown that uric acid is synthesized in the body from the simplest carbon and nitrogen compounds and not exclusively from ingested preformed purines and nucloproteins as had been thought. Therefore, restriction of diet to prevent uric acid stones is not indicated. There is no dietary treatment for cystine stone.

Prevention of uric acid and cystine stone depends on alkalinization of the urine. The solubilities of these substances are considerably enhanced in alkaline urine. The best alkalinizer is sodium citrate as proposed by Albright. Alkaline ash diets are not used because they are unnecessary; patients much prefer the sodium citrate. A rounded teaspoonful (3 or 4 grams) three or four times daily in water has sufficed to keep the *p*H at 7.5. This salt may be bought by the pound and used indefinitely without harm. It is mandatory that patients check the *p*H of their urines to maintain the proper alkalinity. Nitrazine paper will be adequate for this purpose. Occasionally, alkalinization of the urine has merely changed the composition of the stone in a uric acid or cystine stone-former; calcium phosphate and magnesium ammonium phosphate have been precipitated instead. To prevent this a low calcium diet should be given with the alkali.

THE IMPORTANCE OF VITAMINS AND NUTRITION

It is felt by some that vitamin A deficiency is an important factor in stone formation. While avitaminosis and malnutrition probably are factors in stone formation in the endemic stone areas of Asia, it is doubtful that they are important factors in most of North America with its high standard of nutrition. I find no reason to give supplementary vitamins to stone-forming individuals who are eating a well-rounded diet.

HYALURONIDASE

The importance of colloids in increasing the solubility of the stone-forming substances in the urine has been warmly debated. Following Meyer, I have felt that colloids were unimportant. Recently Butt has found that an enzyme, hyaluronidase, injected subcutaneously, produces a protective colloidal effect in the urine. In some instances turbid urine is rendered clear and free from sediment for periods

up to twenty-four hours or longer after injection. There is considerable variation in this reaction; many patients get no clearing of the urine at all and in others it lasts for only a few hours or is minimal.

Butt [12] has reported success with this preparation in a series of patients who had multiple, bilateral, rapidly recurring calyceal calculi. In our slight experience it has failed to prevent bladder calculi in recumbent patients.

We have been studying the fundamentals of the action of this enzyme upon the urinary crystalloids. Thin films of urine dried upon scrupulously clean glass slides are made from the voided urine before injection of the enzyme and at stated intervals after injection until the effect (if any) of the enzyme has disappeared. The slides are then examined upon a special darkfield microscope, called an Ultropak, in which the light comes down from above (instead of up from below as in the conventional darkfield microscope). The effect seen microscopically does not always parallel the gross clearing effect.

In certain cases (fortunately few) the aggregation of crystals is enhanced and stone formation facilitated by the enzyme. This reaction is known as sensitization in colloid chemistry. The enhanced aggregation of crystals in these cases is said to be due to too small a dose of hyaluronidase. Recently, such a case was seen in consultation in which a large cystine stone, completely filling the kidney pelvis, formed in slightly more than two months after removal of a small cystine stone from the same kidney. The patient had received 150 units of hyaluronidase on alternate days for two months. Nephrectomy became necessary. Several other cases in which stone formation occurred rapidly under hyaluronidase therapy are known, apparently due to sensitization.

Hyaluronidase should not be given to stone-forming patients promiscuously. A study of the clearing effect on turbid urine should show a definite and pronounced clearing effect for at least twelve hours after 300 units or more of the enzyme are given subcutaneously as a prerequisite for hyaluronidase therapy. This preliminary test is described by Butt and should be followed carefully.

Dosage requirements will vary greatly. We have found it necessary to give up to 1500 units twice daily in stone cases associated with infection, especially urea-splitting infection, in order to keep the urine clear. One pharmaceutical house (Wyeth) now furnishes an ampule containing 1500 units of the enzyme (cost $3.60 here in Boston now). In many cases even dosages of this size fail to render turbid urine clear. No significant unfavorable reaction to the enzyme has occurred in our cases.

There is some experimental evidence that the sensitization reaction may at times be manifested as the effect of a dose of hyaluronidase wears off. This would suggest that adequate doses of the enzyme should be spaced close enough together to produce a slight overlapping effect.

All stone-formers do not have a turbid urine, however. This makes it difficult to determine the proper dosage of the enzyme. A study of urinary films with the Ultropak microscope is of value in such cases and may determine the proper dosage. I should like to emphasize that I do not regard the Ultropak microscope as a tool of utter precision for this work. Considerable experience is necessary in the use of this instrument in this almost unknown field in order that the findings may be properly interpreted. Extreme variations occur in one and the same urinary film; much of this is probably artifactual and occurs in the making and drying of the slide. However, this technique is the only one of significant value at the present time. In the present state of our knowledge I definitely advise against the purchase of this expensive instrument for a study of this kind.

It is too soon to evaluate Butt's original idea. It takes time to produce calculi and still longer to prove that a particular type of treatment has prevented them. We have had no patient on the enzyme clinically for long periods of time. *What*

has been said does not constitute an endorsement for the use of hyaluronidase to prevent stone. In the present state of our knowledge it would appear that hyaluronidase therapy is not indicated in most stone cases but may be tried in rapidly recurring cases, in those in which intractable infections and other complications exist, and in cases in which our other techniques, after proper application, have failed.

CONDENSED SUMMARY OF MEDICAL MANAGEMENT

For all cases—force fluids

For Calcium oxalate and mixed Calcium oxalate-Calcium phosphate stone

1. Prohibit all dairy products (except butter) and foods with much milk. No milk, cream, cheese, buttermilk, condensed milk, ice cream, cream soups, milk gravies, cheese souffles, Welsh rarebits, and custards. Allow butter ad lib.
2. Keep daily consumption of citrus fruit juices (orange, grapefruit, lemon) and tomato juice to low normal limits. (to avoid large amounts of low alkaline ash).
3. Avoid *excessive* consumption of following items: reasons—high calcium 1, high alkaline ash 2, high oxalate 3. Check thru list with patient to discover any dietary peculiarities—*then forget it.* Insistence on close cooperation on foods in this long and hard to remember list will only lead to in-cooperation. (Where both calcium and oxalate are present it is extremely doubtful that absorption occurs.)

dried fruits — apricots 2, figs 1-2-3, olives 1-2, raisins 2

nuts—1-2-3

Vegetables—asparagus 3, beans 1-2 beets 3, carrots 2, parsnips 2, parsley 1-3, rhubarb 1-3

green leaf vegetables—broccoli 1, chard 2-3, collards 1, greens (beet 1-2-3, dandelion 1-2-3, mustard 1, turnip 1), kale 1, spinach 2-3, water cress 1-2-3.

Sweets—molasses 1-2, molasses candy 1-2, treacle 1-2, maple syrup 1-3, chocolate 1-3

Sea foods—clams 1, oysters 1, shrimp 1, salmon 1, sardines 1.

Remember the above foods are acceptable in moderate amounts

4. Acidification with sodium acid phosphate, 0.6 grams (10 grains) 4 times daily to bring *p*H down to 6.5 or below.

For $MgNH_4PO_4$-$CaPO_4$ stone (alkaline infection)

1. Diet as above
2. Eradicate urea-splitting infection
3. Sodium acid phosphate only if urine can be acidified.
4. Basaljel plus low phosphate diet if urine cannot be acidified (see Chronic Infection with Stone).

For Uric Acid and Cystine stone

1. No dietary treatment (alkaline ash diet unnecessary)
2. Alkalinization with sodium citrate, 3 or 4 grams (1 level teaspoonful) 4 times daily to keep *p*H at 7.5.
3. Low calcium diet. No dairy products (except butter) to prevent calcium phosphate stone in markedly alkaline urine

SUMMARY

A study of urinary calculi and of the stone-forming process by crystallographic techniques (reported elsewhere) has suggested that more emphasis should be placed upon the significance of the nucleus in stone formation and prevention. Normal urine may be able to retain its dissolved salts in a 2 to 4 times supersaturated solution for a time without precipitation occurring if no nucleus is present. In the presence of a nucleus precipitation may occur whenever the saturation point of the urine is exceeded. It is desirable, therefore, to conserve, if possible, this power of urine to hold its salts in solution by preventing nuclei from forming. The nuclei of the majority of calcium-containing calculi (including calcium oxalate calculi) are composed of calcium phosphate. Measures to inhibit the formation of such nuclei may be of value in a stone-prevention program.

The underlying principle of such programs is the prevention of excessive supersaturation of the urine by stone-forming salts. This may be done by increasing the urinary volume, by decreasing the urinary excretion of the offending crystalloid, and by increasing the solubility of the offending crystalloid. The details of the various regimens are presented. Hyaluronidase therapy is also discussed.

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CLINICAL EXPERIENCES WITH TELEPAQUE * †
(A PRELIMINARY REPORT)

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Telepaque, the new cholecystographic medium, when used in accordance with patient preparation and dosage advocated by the manufacturer, produced results, which in our experience, could only be graded as satisfactory. However, the repeated occurrence of certain observations during preliminary trials prompted changes, which in our hands, have produced results better than any other procedure utilizing an orally administered drug. These changes are a radical departure from the advised patient preparation and dosage, and to our knowledge, are different from any other accepted procedures.

The details of the physical properties, experimental data, etc., of telepaque are obtainable from the manufacturer, but for convenience, a resumé of pertinent facts is offered.

Telepaque is an organic compound, containing iodine, the chemical formula for which is 3-(3-amino-2, 4, 6-triiodophenyl) -2-ethylpropanoic acid. Its iodine content is 66.6 percent. Telepaque is insoluble in water, but is soluble in dilute alkali, 95 percent alcohol, and other organic solvents. Intravenously, telepaque is slightly more toxic than other cholecystopaques, but orally the lethal dose is about four times that of the next less toxic. Comparative density studies revealed that 500 mg. of telepaque, per kilo of body weight, produced shadows equivalent to dosages of 1000 mg. per kilo of other orally administered media. This was corroborated by the studies of Morgan and Stewart. [1]

After exhaustive study, Epstein, Natelson and Kramer [2] offered three points, which they believed formed the criteria for a good cholecystopaque.

1. It should be phenolic and fat soluble in order to reach and pass through the liver.

2. It should have a carboxyl group for solubility in bile.

3. Its iodine content should be high for better opacification.

Anyone sufficiently interested in the chemical nature of telepaque will find that it compares favorably with the three points of the above criteria.

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* Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

† Materials for this study furnished by Kenneth M. Smoot, Research Department, Winthrop-Stearns, Inc.

PATIENT PREPARATION AND DOSAGE

For the routine examination, the manufacturer advises a nonfat meal before ingestion of the tablets, a standard dosage of 3 grams (6 tablets), and an absorption period of ten to twelve hours before filming. Patients are advised not to eat or drink during the absorption period. Enemas are advised for the removal of intestinal contents and gas.

In following this procedure, it was repeatedly observed, that gall bladder shadows were only of a satisfactory density and large residual accumulations of dye were consistently seen in the intestinal tract. These have been found in the stomach and at many points beyond. One of the first cases studied revealed no gall bladder shadow at twelve hours, but there was a large accumulation of the medium in the cecum and ascending colon. This patient returned twenty-four hours later for a stomach examination and exhibited a normal gall bladder outline of good density.

After this and other similar experiences, it was thought that patients were not being allowed a sufficient period of time to assimilate and store the dye. It was also possible that the advised dosage was too large, since several cases showed good density gall bladder shadows and large residual intestinal accumulations of dye. Remembering that, in the comparative density studies, telepaque shadows were markedly more dense, it was felt that even though this dosage produced no ill effects on the patient, this large dose just did not appear to be necessary.

If these deductions were correct, then dosage should be reduced, possibly it could be adjusted to a weight scale and the absorption time should be lengthened. If the absorption time was to be lengthened to, say eighteen hours, this would require the patient to abstain from food for two normal meal periods, thus adding the discomfort of hunger to the examination. It was decided that nonfat foods could be allowed in moderation, since they would produce no effect upon the gall bladder. It might also be possible that the normal digestive processes stimulated would enhance the assimilation of the drug.

Adjusting the dosage offered no particular problem. Patients were divided into three groups. Group I included all under 130 pounds and reduced dosage was placed at 1 gram (2 tablets). Group 2 included all patients between 130 and 170 pounds and dosage was fixed at 1.5 grams (3 tablets). Group 3 included all over 170 pounds and dosage set at 2 grams (4 tablets). By the same logic, which suggested this scale of dosage, it is reasonable to suggest that dosage will have to be increased beyond 2 grams for the abnormally large individual. However, to date, several patients whose weight reached or exceeded 250 pounds have been satisfactorily examined with 2 grams (4 tablets).

No specific instructions were issued as to the taking of the drug; patients were simply told that they could take the tablets singly or all at once.

Early in the study, a small group of patients began to show irregular results. This was the borderline group of those just under and those just over 170 pounds. By measurement, it was found that the short squat individual was often thicker through the filming area than the taller, even heavier person. It became immediately apparent that body thickness was almost as important as body weight, and body thickness was incorporated as an additional criteria for this borderline group. Thus, dosage was readjusted and some Group 2 patients were treated as though they belonged in Group 3 and some Group 3 patients as though they belonged in Group 2. After this change it was noted that the density of gall bladder shadows became more uniform.

It was thus that a routine of patient preparation and dosage was arranged. Simply stated it is as follows: Patients are questioned as to diet. If they have been given one, they are instructed to follow it during the day preceding filming. If not, they are instructed to eat whatever they desire. The patient is given the

exact number of tablets according to his grouping and instructed to take them eighteen hours before the filming time scheduled the next day. For the two meals inevitably included during the absorption time, a list of nonfat foods is given, from which the patient may choose his meals. Patients are cautioned as to overeating, especially in the mornings, because of the fat meal they will have to ingest in order to complete the examination. Water and soft drinks are not restricted, alcoholic beverages are.

SIDE EFFECTS

It is not the purpose of this paper to list in detail the number and frequency of side effects resulting from the ingestion of telepaque. Suffice it to say, the infrequency of side effects noted is comparable with other reported studies, namely:

A greater number of patients were asymptomatic.

Diarrhea was markedly reduced. It is to be stated, however, that telepaque does appear to have a definite laxative action. This may often appear as late as thirty-six hours after ingestion of the tablets. Most patients will report a definite change in bowel habit, whether it be a softened, loose, or watery stool, or even an increase in number of stools. Frank diarrhea, however, is rare.

Nausea and pyrosis were reduced. None of the patients examined reported vomiting. Dysuria, headaches, flushing, and other effects were infrequent.

RESULTS

Observations made from the films warrant comment. All known variants of technique were tried with variable results. Reported visualization of ducts with large dosage and short absorption time was unsuccessful in our hands. Patient preparation and dosage as advocated by the manufacturer was only satisfactory.

Following the previously described patient preparation and adjusted dosage, gall bladder shadows have shown greater opacification. This compares favorably with the manufacturer's claims and other published studies. Densitometer readings were not made in this study, but from the films alone, density and sharpness of outline were noticeably increased.

In addition to the denser gall bladder outline, the normal "s" shaped curvings of the neck, the cystic duct and less frequently the hepatic duct were visualized.

Visualization of the ducts has been a more frequent occurrence in the thin and medium individuals and has seldom occurred in the heavier built persons. In the thin and medium built individuals the gall bladder is seen to hang downward toward the right and the ducts course upward then downward toward the left, while, in the heavy individual the gall bladder and ducts follow a more horizontal course. When the thin and medium individuals are positioned for filming it is seen that the gall bladder and ducts are more perpendicular to the examining rays, while in the heavier person the course is more parallel. This condition of habitus alone, added to the poorer quality films obtained in the heavy group forms a reasonable explanation for the failure of visualization of the ducts.

A frequent objection by examiners is that the greater opacification with telepaque obscures small stones. This may be true, but in the questionable cases, a slightly overexposed film should clear up any doubt as to the presence or absence of stones.

It is well to remember that the concentration of telepaque in the gall bladder increases the specific gravity of bile. This is sufficient to cause small stones to float up and sometimes not to be observed if they become crowded in the curvings of the neck. This was beautifully demonstrated in the studies of Kirklin, which he reported in the Rigler lecture in 1952.

Patients, in whom a gall bladder has visualized, often report twenty-four hours later for additional roentgen studies. In all of these, it has been noted that the gall bladder has revisualized even if evacuation has been complete in response to fat meal. This second visualization is

often of satisfactory to good density and often beautifully demonstrates the relationships of the gall bladder especially if the stomach and duodenum have been barium filled.

NONVISUALIZATION

In following the revised patient preparation and adjusted dosage, nonvisualizations began to occur with sufficient frequency to become a disturbing factor, especially when the patient has reported a minimum or no side effects and no residual dye could be discovered in the intestinal tract. It is a well known fact, that a gall bladder will often spontaneously evacuate itself. Whether this has happened during the prolonged absorption period, or telepaque with its known laxative effect, and higher specific gravity may have induced evacuation, or the patient has ingested something which has caused evacuation has not been determined.

These nonvisualizations have been sufficiently frequent to force an addition to the routine of examination. In all cases of nonvisualization the patients were instructed to continue on the nonfat diet they had received. An additional dose of telepaque was given with instructions to take the tablets eighteen hours before refilming the following day. It was felt the additional dose of telepaque was merely a precautionary measure, to replace any of the dye lost through the processes of normal elimination, because experience has shown that gall bladders revisualize as a rule, and this additional dose may be entirely superfluous. Experience to date is too small to be conclusive.

Upon return of the patient the following day, more often than not a gall bladder outline is seen, fat-meal is administered and its function reported. If, however, on the second day the gall bladder remains invisible, it is felt, that after these two attempts, it is safe to report the gall bladder as nonfunctioning.

All of us are aware that telepaque, like all new drugs, is still under observation. Even after a year of trial and several hundred examinations it is felt that standards for examination with this media have not been set, even though it appears to produce results superior to other orally administered cholecystopaques.

CONCLUSIONS

The only conclusions warranted at this time are:—

1. Side effects, from the ingestion of telepaque at the suggested dosage, are so slight as to render this an innocuous procedure.

2. More satisfactory results have been obtained utilizing the eighteen hours absorption period. Shorter times have not proven satisfactory in our hands.

3. It is still a question whether the ingestion of nonfat foods materially interferes with or enhances the routine of examination.

4. In cases of nonvisualization, the additional dose of telepaque, with refilming in twenty-four hours has been invaluable in the determination of the function of the gall bladder.

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DISCUSSION

Dr. Joseph P. Tomsula (Baton Rouge): Drs. Brierre, Hunt and Landry are to be complimented not only for critically assaying but also for tailoring a new product to individual usage. We are too often prone to accept the carefully selective results of larger centers without question or variation until we suddenly realize that our own results do not compare. Patients may or may not be comparable but techniques can and do vary.

Dr. Francis Spencer from San Angelo, Texas, did report dosage fluctuation in the August 1952 issue of Gastroenterology but he did not carry it to the program adherence we have had outlined

this afternoon. Practically all writers comment upon the dye residue within the colon. None appear to regard it as a great hindrance to gall bladder visualization. Yet one can gather that all would feel better if it were not there. This implied wish makes the efforts of our own colleagues even more propitious.

During August and September of 1952, all of our gall bladder patients (40) at Our Lady Of The Lake Hospital were divided so that one received telepaque—the next a double dose of priodax. A maximum of side effects can and do occur with the above priodax regime. By comparison the 3 gram or 6 tablet dosage of telepaque was remarkably free from side effects. Additionally, the gall bladders were much better visualized. We made no attempt to vary from the 6 tablet regime. All our radiographic studies were taken within twelve to fifteen hours following ingestion of the drug. All received a synthetic fatty "meal" after the preliminary films were seen and the postcibum studies were then taken thirty to forty-five minutes later.

From its initial usage through April 1953, 277 of our patients have received this drug. If the gall bladder did not visualize with the use of telepaque, the patient was given a double dose of priodax. We have had none visualize with priodax if it did not do so with telepaque.

It is an unusual occurrence not to see the cystic, and at least a portion of the common duct, in small and average sized patients on the postcibum studies. With large individuals this incidence is reduced but is not always absent. We have also had an excellent correlation of radiographic and surgical findings.

I want to thank Drs. Brierre, Hunt and Landry for a very timely and worthy presentation. Perhaps we should also thank them for proving that we as individuals in the practice of radiology can assay a new product and contribute to the knowledge of its safe everyday usage.

BRONCHOGENIC CARCINOMA A DIAGNOSTIC PROBLEM

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INTRODUCTION

We are all aware that given an early case of carcinoma of the lung one can affect a cure by pulmonary resection. This has been proven many times since Graham's successful resection in 1933. The problem that is greatest is accomplishing a diagnosis early enough to be able to resect with the hope for cure. In this study we propose to analyze the difficulties in arriving at a diagnosis of carcinoma of the lung, and particularly to study this problem in our own community; and, furthermore, to see what can be done to bring these lesions to earlier surgery. The first pulmonary resection for carcinoma of the lung was not done until 1948 in our community. We believe that we have progressed since then and should be able to detect cases earlier. But, can we?

We have tried to be cancer conscious with all of the pulmonary lesions presented to us and have encouraged our colleagues to do likewise. Nevertheless, in our local hospitals the story is identical to that in hospitals all over this country and in other countries all over the world; the incidence is increasing but the resectability and curability rates remain about the same.

There seems to be little doubt that carcinoma of the lung is increasing; in fact, some suggest that it is increasing more rapidly than any other type of cancer. In this country during the decade ending in 1948, deaths from lung cancer increased 144 per cent in contrast with an increase of only 31 per cent in deaths from all types of cancer. The cause of this increase is not a purpose of this thesis and will be left to the centers where large series of cases are studied. At the Hines Veterans Hospital the period covering 1931 through 1936, the percentage of cancer admissions due to carcinoma of the stomach remained relatively stable at about 5 per cent while that of carcinoma of the lung increased 2 per cent to 12 per cent.

The disease is seen predominantly in the male sex, the ratio being approximately 8 to 1, and most commonly in the fifth, sixth, and seventh decades. The locations of the lesion are approximately 50 per cent in the upper lobes. More lesions are seen on the right than on the

* Presented at the Seventy-Third Annual Meeting of the Louisiana State Medical Society, May 8, 1953, in New Orleans.

left which is probably accounted for by the difference in size of the two lungs. Pathologically, approximately one-half of the resected carcinomas of the lung are squamous cell, about one-third are undifferentiated cell carcinomas, and about one-sixth are adenocarcinomas.

The onset of respiratory tract carcinoma is always insidious and misleading as evidenced by one of our cases that began with multiple superficial thrombophlebitis. However, the leading symptom reported in all series is that of cough. We have come to believe that cough is probably the most important symptom, i.e., the persistent cough that remains after an upper respiratory infection in the nonsmoker and the change and increase in intensity in the cough of a smoker. In the Brompton Hospital series the early symptoms in order of the frequency were cough, pain, dyspnea, hemoptysis, febrile illness lassitude and combinations of the six above. The average duration of symptoms is usually from nine months to a year before consulting a physician. The average duration of symptoms in our cases was approximately one year. Hemoptysis is the one symptom which will induce the patient to consult a physician without delay. It would be helpful if all carcinomas of the lung would bleed early, and therefore, cause the patient to seek medical counsel. Unfortunately, as in all other carcinomas, bleeding is sometimes a late symptom or is not seen at all. In our cases about 18 per cent had hemoptysis as an early sign. Early diagnosis of carcinoma of the lung is dependent upon the index of suspicion of the physician. All negative lesions seen on x-ray should be thoroughly investigated. The value of bronchoscopy is limited; approximately 20 to 30 per cent can be seen on bronchoscopy. The number of positive diagnoses can be increased still further with cytological studies of sputa and bronchial washings. The use of bronchograms and planograms often aid the roentgenologists. However, approximately one-third of all pulmonary lesions must be explored without a positive preoperative diagnosis.

CASE REPORTS

Case No. 1: Mr. W. 68 year old white male. Onset was manifested by persistent cough, pain in right chest, and fever. The first x-rays revealed right upper lobar pneumonia. He improved on antibiotics, but his chest x-ray did not completely clear. Bronchoscopy and bronchial washings were negative. He was not offered an exploratory thoracotomy. He returned five months later and upon exploration he was nonresectable.

Case No. 2: E. W. 62 year old colored male. Onset was manifested by cough and fever. The first chest x-ray showed a pneumonitis in the left midlung zone. The radiologist suggested that this might be secondary to a mass lesion. With penicillin the patient improved but did not heed his physician's warning. Four months later the patient was asymptomatic. Routine retirement chest x-ray was taken and a mass lesion was seen in the left upper lobe. With the Broyle's bronchoscope the left upper lobe orifice was now seen to be closed, suggesting a tumor. Bronchial washings failed to show malignant cells. A pneumonectomy was done and in the lumen of the left upper lobe was a squamous cell carcinoma, well localized and obstructing the lumen.

Case No. 3: Mr. S. 58 year old white male. This patient was being followed by means of x-ray because his daughter had recently died of tuberculosis. He denied any symptoms except pain in his right shoulder and arm. X-ray of September 12, 1949, showed some haziness of both apical regions of questionable activity. Films of January 6, 1950, showed increased haziness appearing in the right upper lung field. Films on May 4, 1950, were more definite, and one year later the lesion was quite definite and was still being called tuberculosis with never a positive sputum for acid fast bacilli. In November 1951, we saw the patient for the first time; bronchial washings revealed malignant cells and pneumonectomy was done. Pathological section showed mixed adeno and squamous cell type of carcinoma arising from the right upper lobe bronchus.

Case No. 4: 54 year old colored male, who was followed as a probable tuberculosis case for six months with never a positive sputum for acid fast bacilli. At operation he was inoperable and died three months later of metastasis.

Case No. 5: Mr. W., age 52. Onset of cough, hemoptysis, and a diagnosis of tuberculosis was made without a positive sputum. The patient was treated with streptomycin, etc. One year later bronchoscopy showed marked narrowing of the left main stem bronchus. However, biopsy was negative. The patient refused exploratory

surgery. Eighteen months later the diagnosis was obviously carcinoma; the sputa cytology was positive and the patient had evidence of large nodules in the liver. He died one month later.

The lesson here is never to accept an x-ray diagnosis as an etiological one. X-ray shadows are only suggestive and must be substantiated by laboratory and clinical findings.

Case No. 6: Mr. F., white male, 68 years of age. Presenting symptom was hemoptysis which lasted two or three days. On bronchoscopy a positive biopsy was obtained. Surgery was advised to the patient through the patient's physician. Surgery was refused at this time. The patient returned ten months later and was obviously inoperable.

It is our belief that if properly explained to a patient that he has carcinoma, and that his only hope for survival is through surgery, none will refuse operation.

Case No. 7: Mr. M., age 41. Illness began with abdominal pain, and an emergency appendectomy was done on June 8, 1951. One month later he returned with pleural effusion following upper respiratory infection. Repeated thoracenteses were done and cytological studies failed to make a positive diagnosis. Repeated sputa were studied for acid fast bacilli and tumor cells. The pleural effusion continued to recur, and in November 1951, the patient presented a painful swelling of the right costal margin. On exploration of this lesion the posterior rectus sheath was found to be thickened and hard, and a frozen section showed a squamous cell carcinoma. Upon thoracotomy a tumor was found arising within the right lower lobe with marked spread throughout the pleura and pericardium.

Case No. 8.: Mrs. F., 52 year old white female whose presenting symptoms were fever, malaise, and pain in the left chest. She developed a left pleural effusion which was aspirated and was negative for acid fast bacilli and tumor cells. Her symptoms progressed, and six months later she was admitted to a tuberculosis sanatorium. The x-rays were quite suggestive of tuberculous pericarditis. Repeated laboratory studies failed to produce an etiological diagnosis, and on May 19, 1952, the chest was explored through a trans-sternal incision. The pericardium presented a thick sheath of carcinomatous infiltration. There were firm carcinomatous nodules throughout both lower lobes.

The problem which is presented by cases No. 7 and No. 8 is this: Should unexplained pleural effusions be explored?

Case No. 9: 62 year old white male with severe cough for four or five months and hemoptysis. Positive biopsy at bronchoscopy was obtained. The patient has been well three and a half years now.

Case No. 10: Mr. A., 65 year old white male, who presented symptoms of cough, severe dyspnea, and cyanosis. After work-up a carcinoma of the left lung was suspected but was not proven; however, the patient had too little respiratory reserve to warrant exploration. He was cyanotic lying in bed. Two years later he returned with the same symptoms and an obvious carcinoma with a positive bronchoscopic biopsy. He was operated upon by another surgeon and died during surgery.

The interesting thing shown here was that the lesion could be followed by x-ray for over four years but yet the patient always had too much emphysema, etc. to warrant an operation.

Case No. 11: Mr. L., 50 year old white male who presented himself because of persistent cough. He was told that he had a very long uvula and that it was tickling his throat. A uvulectomy was done. His cough persisted and subsequently he had hemoptysis. He was then sent to a chest center where on work-up the diagnosis of carcinoma of the lung was made and palliative pneumonectomy was done.

Case No. 12: Mr. D., 70 year old white male who had onset of cough and pain in his left chest. He was seen by his physician who made a diagnosis of virus pneumonitis. We saw this patient later; he had many sputa examined for cytology, fungi, and acid fast bacilli. Bronchoscopy and bronchial washings were negative. The chest pain persisted but his x-ray did not change. The patient was scheduled for exploration, but it was cancelled because it was felt that there was insufficient evidence to warrant thoracotomy on a man this age. Two months later a Bucky film showed erosion of the ribs over the site of pain. A biopsy of the ribs proved to be epidermoid carcinoma. The onset is often so insidious that it fools even those who are constantly on the lookout for these lesions.

Case No. 13: Mr. O., 50 year old white male whose complaint was lassitude. He had no cough, had occasional vomiting, and had lost approximately 10 pounds of weight in the past three months. X-ray showed a mass lesion. Bronchoscopy and cytologic study of sputa were negative. On exploration, the hilum was frozen and the lesion was not resected.

The sad feature of this story is that the patient was hospitalized some six months previous to this film for a tonsillectomy. A chest film was not made. Now, however, a photofluorogram of the chest is made routinely on all admissions to this hospital. It is probable that the lesion could have been seen six months earlier and could have been resected at that time.

Case No. 14: 54 years old white male whose presenting symptom was thrombophlebitis of the

left lower extremity in November 1952. He had hemoptysis at this time which was explained on the basis of pulmonary embolus, and quite reasonably so. X-rays of the chest at this time showed increased hilar markings on the left. In March 1953, four months later, the patient was seen as a diagnostic problem because of multiple thrombophlebitis in all extremities, involving both the superficial and deep veins. The repeated hemoptysis had continued and the patient was coughing. Cytology of sputum at this time showed positive carcinoma cells. A supraclavicular node was palpated and proved to be positive on biopsy. X-rays of the chest at this time showed an obvious tumor that in retrospect could be seen in the first films at the onset of his illness.

This case is presented to further substantiate how varied the onset of signs and symptoms of pulmonary carcinoma can be and therefore warrant a complete work-up on all cases.

SUMMARY

From this survey it is obvious that there is a need for education of the public and a greater awareness on the part of the physician regarding the frequency of the disease, its early consideration in any patient with pulmonary pathology, and the necessity for making an etiological diagnosis in all lesions of the lung.

In this group of cases the problems of pleural effusion, persistent chest pain, recurrent pulmonary infection, confusion with tuberculosis without positive diagnosis of either disease, and incomplete work-up as evidenced by the patient with uvulectomy are presented.

In order to discover tumors in the asymptomatic stage and to increase the salvage rate the value of mass chest surveys has been amply demonstrated in the reports of Overholt and co-workers. In their series of 35 malignant lesions found on chest survey all were resectable and 75 per cent showed no evidence of lymphatic spread as compared to Ochsner's cases (that were not found on chest survey) in which 35 per cent were resectable and only 10 per cent had no evidence of spread.

It should be stressed that every male past the age of 40, and especially the heavy cigarette smoker, should have an x-ray of his chest every 6 months. Also a small chest film should be made routinely on every hospital admission, when practicable.

In all patients with unexplained pulmonary lesions, exploratory thoracotomy should be seriously considered.

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OBSERVATIONS ON THE USE OF TELEPAQUE; A NEW CHOLECYSTOGRAPHIC MEDIUM

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GRANVILLE M. RILEY, M. D.

SHREVEPORT

During the past three years there has been considerable interest in the newer radiopaque media for oral cholecystography as evidenced by the large number of reports in the literature concerning the use of priodax, * monophen, † teridax, * and telepaque. ‡ Priodax has been in use since 1941, and was the first oral cholecystographic medium used which rarely caused any unfavorable reaction in the patient. [2] Monophen has been available for general use since 1944, and while it, too, is a satisfactory medium, [3] its main disadvantage is the size of the capsule which many patients find difficult to swallow and less convenient than the tablet form.

* Schering Corporation, Bloomfield, New Jersey.

† National Synthetics, Inc., 270 Lafayette St., New York 12, New York.

‡ Telepaque for this study was supplied by Kenneth M. Smoot, Southern Division, Department of Medical Research, Winthrop-Stearns, Inc., 111 South Jefferson Davis Parkway, New Orleans, Louisiana.

Priodax and monophen contain 51.5 percent and 52.2 percent firmly bound iodine, respectively, and following ingestion from 50 to 95 percent of the chemically unchanged medium is excreted in twenty-four to forty-eight hours, mainly by the kidneys.

Recently two new preparations have appeared on the market, namely: Teridax,[4] which contains 65.5 per cent iodine by weight, and telepaque,[1,3,5] containing 66.68 per cent iodine. Teridax is readily absorbed from the gastrointestinal tract and excreted principally by the kidneys. Radiopaque material in the colon from unabsorbed dye seldom occurs following its use. It is reported to be well tolerated and has a low level of undesirable reactions. In our experience, visualization of the gallbladder afforded by teridax is considerably less than when telepaque is used. The greatest degree of radiopacity of the gallbladder occurs with the use of telepaque in doses of 2 to 3 grams, depending upon the weight of the patient. It is eliminated mostly by the gastrointestinal tract and to some extent by the kidneys, which accounts for the high incidence of unabsorbed dye in the right colon. This rarely interferes with interpretation, however, and has the advantage of positive evidence that the patient has ingested the gallbladder medium as directed. In the rare case in which it does interfere, this can be corrected by the use of a sodium bicarbonate enema, as telepaque is soluble in dilute alkalis, and by compression spot film radiography to be described later.

During the past two years we have used telepaque for examination of the gallbladder in over 300 cases. The first 50 cases were interviewed for any evidence of unfavorable side effects. In every case, the examination was done to rule out or confirm suspected gallbladder disease. Of these first 50 patients, 1 had nausea, vomiting and skin rash several hours following ingestion of the drug; 2 patients had nausea alone; and only 1 patient reported diarrhea. We have been very much impressed by the low degree of toxicity of this drug. It produces excellent visualization of the gallbladder and occasionally demonstrates the biliary ducts. The youngest patient thus far examined by us was 2 years of age and weighed 26 pounds.

Figure 1.—Normal gallbladder showing excellent visualization using telepaque. The common bile duct and a portion of the duodenum are seen medially.

He received 2 telepaque tablets (1 gram) which produced excellent visualization.

The technique of the examination is as follows: A fat-free evening meal is given and after the meal telepaque tablets are swallowed one at a time five minutes apart with sips of water. Others[3] allow the tablets to be taken with the evening meal and report excellent results. The patient is allowed small amounts of water until midnight, then nothing by mouth until the radiographic examination is made. Twelve hours following ingestion of the tablets the first two routine views are taken, one in the posterior-anterior recumbent position and the other in the left anterior-oblique upright position. These films are then developed and viewed before proceeding with the examination. If the gallbladder is well defined with no overlying gas or radiopaque residue in the colon, a fatty

Figure 2.—Excellent visualization of the gallbladder in the posterior-anterior recumbent position. No evidence of calculi in this projection.

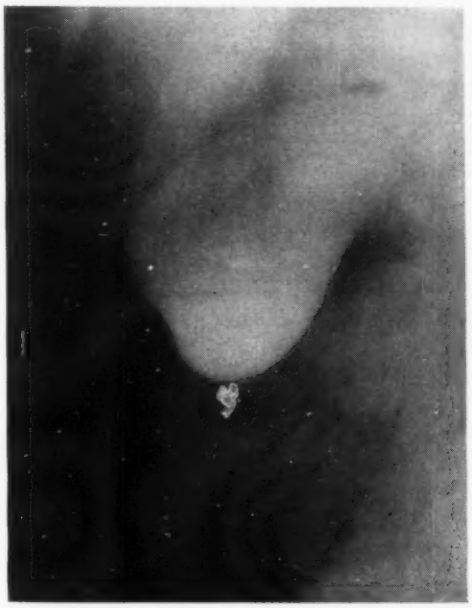

Figure 3.—Same case as Figure 2, taken in upright oblique posterior-anterior projection. Note layer of tiny radiolucent calculi which would have been missed had this position not been used.

meal consisting of 4 oz. cholex (or cholestim) and water is then given and the post cibum film taken thirty minutes later in the posterior-anterior recumbent position.

The reason for using this type of fatty meal is that it does not interfere with barium study of the stomach and duodenum immediately following the cholecystogram. We are of the opinion that the post cibum film, used by many as an index of gallbladder function is of little value since

Figure 4.—Multiple radiolucent filling defects in a well visualized gallbladder. See Figure 5.

many pathological gallbladders will show good response and normal ones fail to respond. Its chief value is that of an additional film which often shows greater dye concentration than is present before a fatty meal is given and occasional visualization of the biliary ducts.

In the event the gallbladder is partially obscured by gas or colon contents, compression spot film radiographs using high voltage (85 KV) in multiple projections are taken. The greater density produced by telepaque makes visualization of the gallbladder by roentgenoscopy relatively simple and high voltage technique allows one to "see through" the gallbladder shadow in

Figure 5.—Upright high voltage spot film radiographs of same case as Figure 4, showing no change in position or filling defects. Diagnosis of polyposis made. Surgical specimen showed polypoid collections of cholesterol adherent to gallbladder wall (cholesterolosis).

the same manner that high voltage technique allows one to visualize polyps in the colon. The routine upright projection is indispensable as numerous cases of small calculi will be demonstrated by "layering" or strata formation in this manner, and will not be visualized in the recumbent position.

CONCLUSION

The increased iodine content of telepaque has made it the most satisfactory cholecystographic radiopaque medium we have thus far used. It is of low toxicity and produces excellent visualization of the gallbladder. High voltage compression spot film radiography, in our opinion, will eliminate the masking effect of increased radiopacity produced by this medium and has the added advantage of displacing gas containing loops of bowel away from the gallbladder.

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SOME USES OF PANTOPAQUE MYELOGRAPHY * †

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NEW ORLEANS

In 1918, Dandy [5] suggested that the spinal cord and its investments could be studied by the replacement of spinal fluid with air. Three years later, French investigators, Sicard and Forestier, [27] had substituted lipiodol for air. Since lipiodol proved to be a mild irritant, a number of other substances were tried. In about 1941, pantopaque was introduced [20, 24] and has become increasingly more popular in the United States. It rarely causes any serious complications, has sufficient density for good contrast, and is of suitable viscosity and specific gravity for even flow within the subarachnoid space. The purpose of this communication is to discuss some of the applications of pantopaque myelography.

PRELIMINARY STUDIES

Preliminary to the performance of myelography, good diagnostic plain films of the portion of the spine to be investigated should be secured in AP, lateral and oblique projections [3, 26]. Careful study of these will provide information regarding the alignment of the spine, and the integrity of the bodies, neural arches and disc spaces. Recognition of congenital anomalies and posterior degenerative spurs is of paramount importance, because

* Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, New Orleans, May 9, 1953.

† Permission for publication of this paper has been granted by the Surgeon General of the U. S. Public Health Service.

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failure to do so may lead to mistaken diagnoses or to operation at the wrong level. Preliminary films should always be compared with the films made at the time of myelograms [13]. Before operative procedure, consultation of the surgeon and roentgenologist to correlate the clinical and roentgen findings is invaluable in arriving at a more accurate preoperative diagnosis and a mutual understanding of significance of anatomical variations.

INDICATIONS

Camp's [3] list of indications for myelography leaves little to be desired. Briefly, he believes myelography is indicated (1) to determine the presence and type of a lesion when clinical evidence is uncertain or indefinite, such as differentiating a tumor from a degenerative disease; (2) to establish the exact level and type of lesion, thereby facilitating laminectomy of limited extent when there is clinical evidence of a lesion; (3) to exclude possibility of multiple lesions when only one is suspected; (4) to determine the cause of symptoms in patients who have had previous laminectomy; and (5) to establish presence or absence of lesions in medicolegal cases. In addition, myelography should be done to rule out an extruded disc in areas before operative bony fusion is performed. Since the procedure is relatively harmless, there seem to be no real contraindications. Malaise, low grade fever, occasional meningismus, and post-tap headaches sometimes occur following myelography but subside promptly.

TECHNIQUE

Accuracy of the procedure is dependent on proper technique. Many methods have been devised, but the one outlined by Wood [26] is highly satisfactory, with slight modification. His method uses a team consisting of a neurosurgeon or surgeon who performs the lumbar tap and other maneuvers requiring sterile technique, and a roentgenologist for the fluoroscopy and roentgenography. Preoperative sedation may be given routinely or reserved for the apprehensive patient. The spinal tap is performed on the fluoroscopic table which should be capable of being tilted either direction at least 45°. The tap is done at least one interspace above the suspected lesion to avoid confusing needle defects in the pantopaque column. Local anesthetic is used to infiltrate the skin and soft tissue at the level of the tap. A No. 18 or 19 spinal needle with a blunt bevel appears to lessen the incidence of subdural injection or formation of bubbles of oil within the subarachnoid space. If multiple attempts to enter the subarachnoid space are necessary it is probably better to postpone the procedure for about ten days because the subarachnoid membrane is almost certain to be lacerated sufficiently to produce subdural collections of spinal fluid, blood, or pantopaque which will interfere with the accuracy of interpretation. When free flow of spinal fluid from the needle occurs, manometric studies may be made if desired. Six to 10 cc. of cerebrospinal fluid are then replaced by 6 cc. of pantopaque for lumbar or dorsal studies, or by 9 cc. for cervical studies. If the oil does not flow into the canal easily, only 1 cc. should be injected until fluoroscopic observation proves that oil passes freely along the subarachnoid space. If the oil remains fixed, it is, most likely, in either the subdural or epidural space and should be withdrawn, if possible, and the examination postponed for about two weeks to allow for absorption of the oil.

After injection of the oil, the stylet is placed in the needle and covered by a sterile dressing. The dressing may be secured by a sterile rubber band. The patient is then placed in prone position on the tilt table, and the foot of the table depressed sufficiently to pool the oil in the cul-de-sac. Careful fluoroscopy is performed, gradually changing the tilt of the table so that the head is depressed and the oil flows upward to whatever level is indicated. When cervical lesions are suspected, care is exercised to keep the head and neck in marked hyperextension to avoid the oil entering the skull. Once oil is in the ventricles, it cannot es-

cape. Routinely, spot films are made in AP and oblique projections at the levels where lesions are suspected clinically or fluoroscopically. Prone lateral films are made with the overhead tube at these levels also. Inspection of the neural canal well beyond the limits of the suspected lesion is always indicated; otherwise, numerous lesions will be overlooked. On occasion where there is complete block below, oil may be introduced by cysternal puncture to determine the length of the lesion.

After films have been inspected and found satisfactory, the dye is pooled beneath the needle fluoroscopically and withdrawn. Repooling and maneuvering of the needle may be necessary, but, usually, almost all the oil can be withdrawn readily. Scrupulous attention to the details of technique and adequate dark adaption of the eyes of the fluoroscopist is essential to successful myelography.

SOURCES OF ERROR

Deviations from a standard technique are the most common causes of mistaken diagnoses of myelographic studies. Some of them have been discussed in the section on technique and will only be mentioned here:

1. If less than 6 cc. of oil is used, [3, 7, 25, 26] skipped areas in the oil column will lead to diagnosis of a lesion where none exists. Recently, some investigators [1, 15] have advocated the use of enough oil to fill the canal completely up to the level of L-1. Their preliminary results have been very promising and further work should be done.

2. Failure to postpone the myelogram after multiple perforations of the arachnoid membrane have been made in an attempt to get the needle into the subarachnoid space causes pseudodefects from spinal fluid leaks or hemorrhage. Sometimes these multiple lacerations lead to a double column [16, 26] of pantopaque in both the subdural and subarachnoid spaces which makes film analysis very difficult.

3. Myelograms following a spinal tap, done within the previous ten days, may show needle defects [16].

4. Failure to examine the spinal canal well beyond the area of suspected lesion, or failure to rotate the patient into the oblique positions, may result in missing small or multiple lesions [2, 12, 26].

5. A professional source of error is the failure to inspect preliminary films for anatomical variations or pathological abnormalities [3, 26]. Six lumbar vertebrae, spondylolisthesis, kyphosis, posterior degenerative spurring, can and do alter the findings. Careful consultation between surgeon and roentgenologist often results in avoiding unnecessary operations or operations at the wrong level.

6. Previous operations on the spine or previous myelograms may confuse the findings due to change in oil column secondary to operative defects or arachnoiditis [3, 4].

7. Unusually narrow or short neural canals may appear normal where laterally extruded discs do not impinge upon them [2].

ANATOMY

Analysis of myelographic findings is based on a thorough understanding of the anatomy of the neural canal and its contents [25]. The walls of the neural canal are composed of the bodies and neural arches of the vertebra and their connecting ligaments. Within the canal are a series of long cylindrical compartments. The spinal cord itself lies suspended in the middle from the hind brain and from denticulate ligaments. The cord is covered by a thin membrane—the pia mater. Two other thin membranes lie between the pia mater and the bony canal. The most central of these is the subarachnoid membrane and the outer one is the dura mater. Between the dura and the bony canal is the epidural space which is filled with connective tissue, blood vessels, and lymphatics. Between the dura and the arachnoid membrane is a very thin potential space called the subdural space. It contains a thin film of cerebrospinal fluid. Between the arachnoid membrane and the

pia mater is the subarachnoid space filled with cerebrospinal fluid. The spinal cord itself extends from the hind brain to the level of L-2 where it is divided into a number of spinal nerve roots which emerge from the spine at lower levels through the intervertebral foramena. Above this level the spinal nerves branch off in pairs to pass laterally out of the spinal canal. The canal is widest in the cervical region and narrowest in the dorsal area.

NORMAL MYELOGRAPHIC FINDINGS

Normal myelograms reflect findings that one would expect from the anatomy. The oil column ascends with smooth borders along the neural canal with short branches flaring smoothly and symmetrically outward along the nerve sheaths as they depart from the canal. The spinal cord can often be seen in the central portion of the column. Differences in size of the various portions of the spinal canal and cord are reflected on the films. The cul-de-sac is variable in its shape and how far distally it extends. This fact sometimes leads to diagnostic errors if it is not fully understood.

LESIONS REFLECTED BY MYELOGRAPHY

Lesions of the neural canal can be divided into three main types; medullary (involving the spinal cord), extramedullary intradural (involving the dura or arachnoid membranes), and extradural (involving the epidural space). The findings in each instance are those that could be predicted from a knowledge of how the lesions alter the normal anatomy. Brief discussion of the main lesions of each type will be presented along with a description of the typical myelographic findings.

Intramedullary lesions represent about 10 per cent [26] of all the neural canal tumors. Most of them are gliomas, about 70 per cent of which are ependymomas and most of the remainder astrocytomas. Most ependymomas are slow growing and may become so large that they produce pressure atrophy of the nearby neural arch. On the plain film, the interpedicular measurements may be increased and the posterior border of the vertebral bodies scalloped. The tumor may extend along the cord for a distance of several segments. Astrocytomas are usually smaller and more rapid growing. Myelography reflects the tumors by showing an expanded cord [3, 12, 26]. In the cervical and dorsal areas, frequently it may completely obliterate the dural sac so that the pantopaque tapers away from the midline bilaterally. Cauda equina tumors may completely block the canal and only a smooth border be presented from below. Smaller ones reveal only irregular filling defects among the nerve roots. Other tumors which are occasionally found within the cord are oligodendromas, hemangiomas and hemangioblastomas, fibromas, lipomas, dermoids, terratomas, etc.

Extramedullary intradural tumors constitute about 60 to 70 per cent of the neural canal tumors [26, 27]. They are about equally divided between meningioma and neurinomas [6]. Meningiomas are most frequent in the dorsal cord and more frequently in females. Rarely do they erode the bone or produce sufficient calcium to show on the plain films. These are firm tumors, apparently arising from the dura along a broad base and indent and compress the cord. This is reflected myelographically as a smooth indentation on a compressed cord. The change is readily recognized on the PA film when the tumor is laterally placed or on the lateral projection when it is anterior or posterior. Below the level of the cord, the nerve roots are displaced. If complete obstruction is encountered, the tumor is well outlined because of the intimate contact with the oil.

Neurinomas arise from the nerve sheath and are seen in any portion of the cord [26, 27]. They are seen in younger patients than are meningiomas and in about the same proportion in both sexes. Bone changes are more frequent in neurinomas than meningiomas but do not occur until quite late. Some of these tumors pass through the intervertebral foramen into the paraspinal area. Thus a paraspinal

mass and an enlarged intervertebral foramen are typical findings when present. Myelographic findings are essentially the same as those of meningioma.

Other tumors in this group include neurofibromas, dermoids, lipomas and metastatic gliomas.

Extradural tumors are chiefly malignant, the primary ones arising from any of the extradural components or from the nerve roots outside the dura. Metastatic extradural tumors are more common, however, and arise chiefly from the breast, bronchus, or lymphomas. Some of them spread directly from bone metastases. The myelographic findings usually are a long, extensive, smooth indentation passing over several segments with the greatest indentation at the midportion of the defect. If there is complete obstruction, the subarachnoid space tapers off to the point of obstruction. Benign extradural tumors present essentially the same findings.

RUPTURED INTERVERTEBRAL DISC

One of the most controversial topics in myelography is that of ruptured intervertebral disc. Many [9, 17, 18, 21] believe that clinical diagnosis of disc is more accurate than myelography. Estimates as to myelographic accuracy vary from about 60 to 95 per cent [14, 18, 21, 22, 26]. With accumulating experience and with strict attention to the technical part of the examination during the past ten years, myelographic accuracy has gradually risen with both the so-called "false positives" and "false negatives" diminishing. Shipman and Vogel [22] reported 92.7 per cent accuracy in a series of 85 consecutive operatively proved cases using 6 cc. of pantopaque. A previous series of 74 performed with only 3 cc. was about 84 percent accurate.

Study is now being carried out using first 3 cc. and then 6 cc. on the same patient at the same examination to see if this percentage difference is sustained. The surgical member of the team found it was occasionally difficult to determine the preoperative clinical diagnosis from the chart. He stated that when disc was suspected at myelography and not immediately found at routine laminectomy, it was sometimes found after more extensive removal of lamina. It appears that clinical findings and myelography are a supplementary, not a competitive, means of diagnosis. Need for teamwork, therefore, has been accepted at some of the larger neurological centers where myelography is always included in cases of suspected protruded disc. [10, 14, 19]

The protruded disc usually produces a localized indentation in the oil column at a disc space. The canal itself may appear normal but a variation in the symmetry of filling of the neural sheaths suggests a protruded disc. Very minor changes in the column must be carefully correlated with clinical and plain film findings. Large discs show ventral defects on the lateral film. The main differential diagnosis in extruded disc is that of posterior degenerative spur of the vertebral body. It also creates a localized ventral indentation at the disc space level. Careful study of the myelographic and plain films will aid in differentiation except where both lesions are present. Multiple discs are said to be present in from 2 to 10 per cent of cases [3, 25].

Occasionally, postoperative cases or cases in which previous myelograms have been performed are referred for myelography. Some of these have arachnoiditis following the previous procedures. Myelographic findings are usually characteristic, forming irregular distribution of the pantopaque in multiple pockets in the involved areas.[3]

Many other lesions can be demonstrated by myelography but will not be discussed. Findings are about what one would expect in varicose veins of the cord, cord atrophy, Arnold-Chiari syndrome, meningoceles, and epidural infections.[26]

SUMMARY AND CONCLUSIONS

1. Pantopaque myelography has been progressively more popular and increasingly more accurate since its introduction in 1941.

2. Accuracy in diagnostic analysis is largely dependent upon strict adherence to

meticulous technique and skilled teamwork in performance of the examination.

3. Insufficient pantopaque and multiple lacerations of the arachnoid membrane appear to be most frequent errors in technique contributing to inaccurate diagnosis.

4. Careful correlation of anatomical and pathological considerations with clinical and myelographic findings leads to more accurate diagnosis.

5. Intramedullary, extramedullary, intradural, and extradural lesions have fairly characteristic myelographic appearances and can often be differentiated from one another by careful analysis of the fluoroscopic and roentgenographic evidence.

6. Clinical and myelographic means of diagnosing protruded intervertebral discs are complementary and their combined use will increase preoperative diagnostic accuracy.

7. Myelography can be well over 90 per cent accurate in diagnosis of protruded discs.

8. Further study of new techniques and ideas will doubtless increase myelographic accuracy.

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DISCUSSION

Dr. M. D. Teitelbaum (New Orleans): Dr. Shipman's discussion of pantopaque myelography has been most thoroughgoing and reflects not only his interest as a student of the subject, but also his large personal experience. He has stressed the point that myelography is a combined operation requiring correlation of the neurosurgical, orthopedic, and roentgen observations and has indicated that with greater experience and improved technique his diagnostic errors have dropped from 16 to approximately 7 per cent. My concern is with that last 7 per cent.

Myelography is one of the large number of radiological procedures which attempts, by employment of contrast media, to visualize lesions which are not directly demonstrable on plain radiographs. Historically, all such procedures become increasingly perfect as they are more widely employed, until finally, some general agreement regarding optimum types and quantities of contrast material and the most rewarding techniques is reached. There remains, however, for each method a hard core of lesions which are so small or so situated that they cannot be recognized by the most careful

and elaborate search. These represent the ultimate, irreducible source of error of that method.

The limitations of myelography are physical with respect to the contrast material and anatomical with respect to the so-called dural sac. No nonabsorbable, nonmiscible, oily opaque material can be considered ideal. However, air myelography is difficult because of the low grade of contrast and the water-soluble medium used by the Scandinavians (skiodan) requires preliminary spinal anesthesia and is not altogether innocuous. Thorotrast is radioactive and should not be used. Consequently, pantopaque, in this country at least, is considered the best of the available contrast substances.

The dural sac, and this part of the discussion refers chiefly to the diagnosis of posterior herniation of the intervertebral disc substance, exhibits considerable anatomical variability which has a direct bearing on the diagnostic accuracy of myelography. The canal varies from 10 to 20 mm. in width and may extend caudally to any level between L-5 and S-4. Its end may be tapering and narrow or wider and well rounded. Furthermore, the axillary pouches differ in length and size and are not necessarily symmetrical. It is obvious that diagnostic accuracy, all other factors being equal, will be higher with long, wide canals which have well formed axillary pouches than when the dural sac is short and narrow and the pouches are small and nearly filled by the substance of the efferent nerves. Epidural and subdural instillation of contrast material appear to offer certain theoretical advantages in extending the scope of the examination, but practically are inferior to myelography (filling of the subarachnoid space) and these techniques have been abandoned. Recently, direct instillation of an absorbable medium into the intervertebral disc itself has been advocated. The discograms unquestionably visualize ruptures of the disc substance into the intervertebral foramen and even at the point where the ganglia and nerves cross the disc outside the foramen. This method also would differentiate posterior bulging of the disc substance with an intact annulus fibrosus from true central posterior herniation. This differentiation is exceedingly difficult by myelography and represents one of the major shortcomings of the method. However, discography is at least potentially dangerous in that it may introduce infection and might possibly injure the posterior portion of the annulus fibrosus so as to permit a subsequent posterior herniation of the nucleus pulposus. Furthermore, multiple disc punctures would be required if more than one level is to be investigated, and tumors of the cauda equina which might simulate herniation of the intervertebral disc substance would be completely overlooked.

Pantopaque myelography, with due regard to its intrinsic shortcomings, is the best available method for demonstration, localization, and differentiation of mass lesions arising from the spinal cord, cauda equina, intervertebral discs and juxtacanalicular portion of the vertebral column. The error will be from 5 to 10 per cent in skilled hands and will approximate 25 to 30 per cent when the study is carried out by casual, inexperienced observers or without proper correlation of the data.

The Journal of the Louisiana State Medical Society

Established 1844

Published by The Journal of the Louisiana State Medical Society, Inc. under the jurisdiction of the following named Journal Committee:

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SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

The Journal *does not hold itself responsible for statements made by any contributor.*

THE LOUISIANA STATE MEDICAL SOCIETY IS OPPOSED TO ANY LEGISLATIVE ACT WHICH WOULD LICENSE CHIROPRACTORS

Since 1922, the Louisiana State Medical Society has successfully opposed the passage of any bill in the Legislature of the State of Louisiana which would give a license to chiropractors. It is anticipated that such a bill will be presented this year, and judged by the information received from reliable sources, various efforts have been and will be made by the chiropractors to secure its passage.

The Louisiana State Medical Society, as a whole, and the State Board of Medical Examiners are unalterably opposed. They have no intention of accepting a cult which denies that germs can be the cause of illness, who believe that all pathology can be cured by pushing the spine back into line with their hands. They are equally opposed to the acceptance of any group who practice medicine without proper training.

The standards of medical education have been raised continually in the last fifty years through the collective efforts of organized medicine over the nation, and in this task the contribution of the Louisiana State Medical Society and the State Board of Medical Examiners has been substantial. The Medical Practice Act of Louisiana has secured innumerable benefits for the public and for the practitioners of Louisiana. It has been used as a model for the other States, and our Society will continue to make strenuous efforts to prevent its being adversely altered.

Four states do not legalize chiropractic in any form. They are Louisiana, Massachusetts, Mississippi, and New York. To these may be added, so far as the practical effect is concerned, New Jersey, and Indiana, in which the chiropractor must pass the same Board as qualified physicians. Just over a year ago, the physicians of New York successfully resisted the efforts of the chiropractors to become legalized there.

The contention of the misguided persons who advocate chiropractic that as a free American citizen everyone has a right to the selection of a doctor of his choice is not adequate reason for allowing improperly trained persons to practice medicine. Equally fallacious is the statement that the practice of chiropractic is not the practice of medicine. It is common sense that those who treat the sick are practicing medicine. The Supreme Court of Louisiana has ruled that the practice of chiropractic is the practice of medicine. The question of "Am I my brother's keeper?" was raised and answered in the

scriptures centuries ago. It is being answered again in our own times in the general trend of social legislation and by such governmental policies as have established Boards of Examination and Licensure for all the professions and many of the trades. In the complicated structure of modern society the community acknowledges its obligation, and protects its members by the establishment of licensing boards for the professions.

The argument that the chiropractors are among us, in spite of our law, is still no argument to legalize an inadequately trained person who calls himself a doctor. Although there have been some twenty-four convictions of chiropractors who practiced medicine without a license in the State in the past 2 years, there are many still with us. It is obvious, however, that the mere fact that a law does not prevent crime is no reason for allowing crime to go unhampered by legal restrictions. The anticipation is that if they acquired a license the so-called "pure" chiropractors would soon be followed by the "mixers," who, in turn, will attempt gradually to encompass the whole field of medicine.

The opposition of organized medicine to the chiropractor is not based on fear of competition. It is based upon the desire to have medicine practiced on the highest possible plane by adequately trained physicians who realize and accept the grave responsibility involved in the care of human illness. In organizing the opposition to the chiropractic bill, it is recognized that the fight cannot be won in Baton Rouge, but it must be won at home. Every man in the Legislature is receiving literature and personal representations urging his acceptance of the bill. It becomes the duty of every physician to oppose these ideas with the facts. Each doctor should see his representative and senator. He should explain the situation to his patients, who, in turn, will express their opposition to any bill leading to the lowering of the standards of medical practice. After all, the fight is being made in the community interests. Let the members of the community know in what way their interests are at stake.

The presentations on this subject are most timely before civic bodies, service clubs, chambers of commerce, and luncheon clubs. Brochures with adequate data are available at the offices of the Parish and State Societies.

The legislator must be put in the position where he can ignore the vociferous minority and vote for the community's ultimate best interest.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

DANGER AHEAD

We will soon be faced with our usual fight in the coming legislature to defeat the chiropractic bill which comes up for enactment every two years. It will be absolutely necessary prior to (NOW) and at the next session to employ every means at our command, to defeat this bill, and render to our legislators and citizens every assistance possible to show and convince them that the claims of these chiropractors as to their educational qualifications and their claims of curing every disease from which human beings suffer, by adjustments of the spine are without foundation. You, as medical men, know their claims are false, but the unsuspecting public unfortunately does not, and is too frequently led astray by their claims of cures, and are finally caught in their alluring traps. We, as scientific medical men, are not concerned personally about

how many cases they have and are not jealous of them in any way. However, when the Louisiana State Board of Medical Examiners issues to us a license to practice, it carries with it an obligation to look after the health and physical welfare of our citizens and to protect them against all quacks, charlatans and cultists who might come into our state to force their wares on our good people. A Medical Society has among its duties not only the promotion of those practices which advance the art of healing the sick, but also the elimination of those which do harm to the patient, because they are based upon false and unscientific conclusions. It is illegal for these chiropractors to practice in our state as declared and upheld by the State and United States Supreme Courts. These Courts have so decreed that this wholly unprepared group of individuals, by education and training, and not qualified, have not been licensed to practice medicine in Louisiana. The Supreme Courts have further decreed that they (the chiropractors) are practicing medicine and are not licensed to do so by the State Board of Medical Examiners, the only group legally qualified to issue a license to practice medicine in our State. These chiropractors know they are practicing medicine illegally and continue to do so, with as little regard for the Courts and laws of our State, as they have for the unfounded and ridiculously impossible claims they make of curing all diseases by manipulation of the spine and releasing a supposedly pinched nerve. If it were not such a serious thing for our citizens, who fall into their hands, it would be quite laughable to all intelligent people to believe they could, by releasing a pinched nerve, cure diseases like smallpox, yellow, malarial and typhoid fevers, diphtheria, tetanus (lockjaw), pneumonia, tuberculosis, diabetes, appendicitis, gallbladder disease, nephritis (Brights disease), cancer and polio, and many others too numerous and absurd to mention. How any conscientious man or woman with a fair degree of intelligence could believe these chiropractors could cure the above group of diseases is beyond comprehension.

These chiropractors would have us believe that when an epidemic disease spreads among the people of a community, that the vertebrae of all the sick slip out of position at the same time, and begin to pinch the nerves.

If adjustments of the spine cure disease, why don't we have chiropractic Veterinarians?

It is said that in the year 1885, the City of Davenport, Iowa, lost one of its best grocers and fish dealers, when D. D. Palmer, said to be the discoverer of chiropractic, closed his store, to take up magnetic healing. In 1895, he claimed to have cured a colored janitor of deafness by adjusting a bump on the man's back. It is said that this is how chiropractic was born.

It is also said that B. D. Palmer, son of D. D. Palmer, and owner and President of the Palmer School of Chiropractic, addressing a Chiropractors' convention in 1920 said: "Our school back in Davenport is established on a business and not a professional basis. It is a business where we manufacture chiropractors. They have got to work just like machinery. A course of salesmanship goes along with their training. We teach them the idea and then we show them how to sell it."

If the chiropractors are so anxious to practice their cult in Louisiana, why don't they go to a recognized Grade A medical school and secure an M. D. degree? They could, then, take the examination given by our Board of Medical Examiners and, if successful in passing, would be issued a license to practice medicine in Louisiana, and would then be recognized as upright and law abiding citizens. They would not then, have to break the law to practice medicine. No, the chiropractor does not want to do this. He would prefer to have the legislature create a Board for them and come in through the back door under the "Grandfather Clause", rather than be licensed on his merits, qualifications, educational attainments, medical ex-

perience and scientific training. We M.D.'s have to meet these requirements. Why should not the chiropractor be required to meet the same qualifications?

We understand that once they have their own board and license all who apply in addition to those who come in under the "Grandfather Clause", begin to do surgery, administer drugs, reduce fractures and deliver babies. This is what they are doing in other states. Why should we expect anything different of them in Louisiana, if they are granted their own Board by the legislature? We contend as do the Supreme Courts that they are illegally practicing medicine and should pass the same licensing Board, exacting the same requirements and qualifications required of all M. D.'s. The regulation of the practice of the healing arts is not undertaken by the State of Louisiana for the protection of our doctors, but to safeguard and protect the welfare of the people.

We do not want to lower the standards of our medical services to our citizens; the kind of medical services that over the years have partially or completely eliminated from our midst many of the most dreaded diseases like yellow fever, bubonic plague, typhoid fever, diphtheria, smallpox, and many others which were nightmares to our parents and grandparents.

When our physicians, the allopathic group of practitioners, are required to meet the high standards of requirements exacted of them by the rigorous curriculum in our accepted Grade A medical schools, and when our State Board of Medical Examiners demands of all who apply for examination that they meet these qualifications in education and training, it is not hard to see and understand why the American people, including the citizens of Louisiana, are getting the best medical service furnished any nation in the world. Taking these facts into consideration, it is easy to explain, and should be very convincing to the most unsuspecting and doubtful sector or groups of our population when it is known that this kind of service has been responsible for the extension of the longevity of life from 34 years in 1908 to 69 years in 1954. Do you think the chiropractors with their poor educational qualifications and limited training could have accomplished this record?

All members of our profession should immediately contact their senators or representatives personally, and have their lay friends do likewise, enlisting their influence and support in defeating the chiropractic bill which is sure to be introduced at the next session of the legislature. The legislature convenes during the first few days of May so you have only a short time in which to contact your legislators. We owe it to the citizens of our state to defeat this bill since we are held responsible for their health and wellbeing. They look to us for protection. Don't let them down.

———o———

NINTH NATIONAL CONFERENCE ON RURAL HEALTH BAKER HOTEL, DALLAS, TEXAS MARCH 4-6, 1954

For the ninth straight year the Rural Health Council of the AMA has held its Rural Health Conference and proved that meeting with the lay public is good public relations for the medical profession.

This year's theme "Let's Put More 'U' in Community" was demonstrated in each assembly. Since Dr. F. S. Crockett was on a world tour, Dr. Carl S. Mundy acted as the presiding officer. The first morning was given over to the Rural Health Committees of the various states, and the rest of the Conference was opened to everybody. There was an attendance of approximately 500 people, and because of weather conditions many more could not reach Dallas. This year's plan of having one general speaker and a panel of four or five specialists in their own field worked out beautifully. Dr. J. P. Schmidt, Professor of Rural Sociology at the University of Ohio, was a master on handling the

question and answer period. Such outstanding speakers as Drs. Charlotte Benson of Raleigh, N. C., John B. Youmans of Nashville, Tennessee, and Lambert Schultz of Chattanooga, Tennessee, were excellent in their respective roles. The discussion periods were participated in by all present, and no one felt like he had been left out when the Conference was over. All in all, your Committeemen, Dr. M. C. Wiginton and I, were well pleased.

J. P. SANDERS, M. D., Chairman,
Committee on Rural and
Urban Health.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SOUTHWEST ALLERGY FORUM

The Southwest Allergy Forum will hold its fifteenth annual meeting at the Roosevelt Hotel on May 9-10-11.

Allergists from all sections of the United States will attend and an outstanding program has been arranged. Members of the Tulane and LSU Medical Faculties will participate. Copies of the program will be mailed to all members of the Louisiana State Medical Society.

EXPERIMENTAL POLIO VACCINE

Eli Lilly and Company has begun production of an experimental polio vaccine for mass immunization trials to be conducted by the National Foundation for Infantile Paralysis.

Eugene N. Beesley, company president, said the vaccine will be produced for the National Foundation on a nonprofit basis.

"We welcome the opportunity," said Beesley, "of co-operating with the National Foundation by producing material for the clinical trial of the polio vaccine. Our experience in biological manufacturing and in tissue-culture procedures has enabled us to start production promptly."

He emphasized that all the vaccine to be made by Lilly will be for the specific purpose of the mass clinical trials, and its distribution will be under the complete control of the National Foundation for Infantile Paralysis.

PILLS WON'T REDUCE WEIGHT—ONLY CONTENTS OF POCKETBOOK

"Sugar and spice and everything nice"—that's what most reducing nostrums are made of.

Fleecing the fat is the sole objective of most of the patent medicines and fancy devices promoted today for "slenderizing," according to Dr. Max Millman, Springfield, Mass.

Such medicines and devices are inert, useless, ineffective and a waste of time and money. People using them are looking for a short cut to weight reduction—a thing which does not exist—Dr. Millman wrote in a recent Today's Health magazine, published by the American Medical Association.

Usually such nostrums contain only syrup, flour, sugar, powdered milk, powdered eggs, vitamins, minerals and flavoring agents Dr. Millman said, adding:

"Another word to describe the reducing nostrums of today is placebo, defined in the medical dictionary as a 'make-believe medicine given to please or gratify the patient.' They are like so much sugar, salt or other foodstuff having no pharmacologic or medicinal value whatever. They do not diminish the appetite; they do not 'burn up' fat; and they do not cause weight loss."

"The regulation and restriction of food intake and the cultivation of better eating habits still form the basis of all effective and permanent reducing."

DRIED BLOOD PLASMA AIDS IN TREAMENT OF BED SORES

Dried blood plasma and Peruvian balsam, in the form of a paste, have produced striking beneficial effects in the treatment of ulcerated bed sores, it was reported in the October 31 Journal of the American Medical Association.

Preliminary studies showed that the preparation caused rapid healing, according to Drs. A. Bernice Clark and Howard A. Rusk, New York. A striking effect was noted especially in those ulcers in which there was dead tissue and infection. In such cases, the paste's action in removing all dead tissue and infection from the wound was most significant, the doctors added.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1954 ESSAY CONTEST

The Thirteenth Annual Essay Contest of the Mississippi Medical Society will be held in 1954. The Society will offer a cash prize of $100.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificate of merit may also be granted to the physicians whose essays are rated second or third best. Contestants must be members of the American Medical Association who are residents and citizens of the United States. The winner will be invited to present his contribution before the 19th Annual Meeting of the Mississippi Valley Medical Society to be held at Chicago, Sept. 23, 24, 1954, the Society reserving the exclusive right to first publish the essay in its official publication—the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5000 words, and must be received not later than May 1, 1954. The winning essays in the 1953 contest appear in the January 1954 issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.). Further details may be secured from: Harold Swanberg, M.D., Secretary, Mississippi Valley Medical Society, 209-224 W. C.U. Building, Quincy, Ill.

BOOK REVIEWS

Physical Examination of the Surgical Patient; J. Englebert Dunphy and Thomas W. Botsford Philadelphia, Pa., W. B. Saunders Company, 1953, Pp. 326, Illus. Price $7.50.

This book reinforces the great importance of the examination of a patient, utilizing the simplest means, namely, the senses of the examiner. Its entire emphasis is upon surgical conditions.

Some of the outstanding features of the text are its drawings of the locations of possibly palpable lymph nodes, a complete listing of the usual findings in the most common surgical conditions, a separate chapter on the abdomen of infants and children, and excellent coverage of the examination of the injured patient.

The volume is a useful compendium of the most valuable means through which a surgeon can arrive at a diagnosis. It is clearly written and presented. The illustrations are well drawn and well printed. Therefore, I believe this book will be welcomed by teachers and students.

PAUL TRAUTMAN, M. D.

Syphilitic Optic Atrophy; by Walter L. Bruetsch, M. D., Springfield, Ill., Charles C. Thomas, 1953. Illus. Pp. 138. Price $5.50.

In this monograph the author reviews the pathogenesis, clinical features and treatment of this condition, which according to statistics causes more than 20,000 cases of blindness in the United States.

From a complete histopathologic study of the optic pathways and neighboring structures in cases of the three main types of neurosyphilis (general paralysis & taboparalysis, meningovascular syphilis and tabes) the author concludes, in accordance with the opinion of Leri and Stargardt, that there is no significant difference in the pathology and pathogenesis of these types and that the so-called primary syphilitic optic atrophy is the degeneration of the optic nerve fibers which is always caused by an inflammatory process within the nerve itself, mainly situated in its intracranial portion and in the chiasm. Dr. Bruetsch stresses the importance of an early diagnosis by the correct search and interpretation of the clinical findings (pupillary abnormalities, visual field defects, pallor of the disc, etc.) because only if the proper treatment (malaria, penicillin) is initiated before the degenerative changes are advanced can blindness be prevented.

BERTHA R. NOBLE, M. D.

PUBLICATIONS RECEIVED

Appleton-Century-Crofts, Inc., N. Y.: Holt Pediatrics, by L. Emmett Holt, Jr., M. D., and Rustin McIntosh, M. D. (12th Edit.).

Ejnar Munksgaard, Copenhagen, Denmark: Acute Anuria, by Claus Brun.

Philosophical Library, N. Y.: Thoughts About Life, by Felix Friedberg; The Jealous Child, by Edward Podolsky, M. D.

W. B. Saunders Co., Phila.: The Hepatic Circulation and Portal Hypertension, by Charles G. Child, III, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: Acute Renal Failure, by Arthur Grollman, Ph.D., M. D.